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Effects of Intravenous Infusion of Glucose and Pancreatic Glucagon on Abomasal Function in Dairy Cows

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Holtenius K, Jacobsson SO, Holtenius P: Effects of intravenous infusion of glucose and pancreatic glucagon on abomasal function in dairy cows. Acta vet. scand. 1998, 39, 291-300. – Four cows of the Swedish red and white breed fitted with a cannula in the abomasum were used in 2 experiments. In experiment I glucose (4mg/kg bw/min) was infused intravenously for 60 min after an initial control period, without infusion, of 60 min. The turnover time of abomasal fluid was calculated using Cobalt-EDTA as fluid marker. The frequency and amplitude of the abomasal pressure changes were registered during the experiment. The plasma level of insulin and glucose was also registered during the experiment. Due to the glucose infusion plasma glucose increased with about 4 mmol/l. The elevated plasma level of glucose induced a pronounced release of insulin. The turnover time of abomasal fluid increased from 15.7±1.2 to 27.8±3.5 min ($p<0.01$) during the glucose infusion. The mean amplitude of the pressure changes showed a more than twofold increase ($p<0.05$) during glucose infusion as compared with the control period but there was no difference in the frequency of the changes. In experiment II there was a similar experimental set-up with the exception that pancreatic glucagon (30pg/kg bw/min) was infused instead of glucose. The glucagon infusion induced a release of endogenous glucose which in turn increased the plasma level with about 3 mmol/l. The plasma level of insulin rose to about the same extent as during the glucose infusion in experiment I. The turnover time of abomasal fluid was delayed from 15.4±1.7 to 34.8±1.9 min ($p<0.001$). There were no significant effects of the glucagon infusion on the frequency or the amplitude of the abomasal pressure changes. The results of the present study indicate a disturbed abomasal function in cattle with hyperglycaemia. It remains to be investigated if it is a direct effect of the hyperglycaemia or if it is secondary to the elevated insulin level.

laparotomy; emptying rate; amplitude; frequency; glucose; hyperglycaemia; insulin.

Introduction

Left abomasal displacement (LDA) has been recognised since the 1950's in dairy cattle in increasing incidence. Most cases of LDA are seen during the first 4 weeks after parturition in high producing dairy cows. It appears to be generally accepted that an atony or hypotony and hence a decreased abomasal emptying rate results in an accumulation of gas and fluid in the abomasum. The gas accumulation can result in a movement of the abomasum upwards to the left between the abdominal wall and the rumen. (for reference see review by *Geishauser,* 1995). However, the mechanisms regulating abomasal emptying is still unclear. The inflow of digesta into the abomasum occurs constantly and the

outflow to the duodenum is a nearly constant process. The regulation of the outflow does not appear to be under tight feed-back control from receptors in the duodenum, as in monogastric species and in pre-ruminants (*Gregory & Miller,* 1989). A large number of factors have been shown to play a role in the aetiology and pathogenesis of LDA and many of the factors are obviously related (*Geishauser,* 1995). High energy rations during the dry period and high concentrate diets early post partum seem to be among the factors that predispose to LDA (*Coppock et al.* 1972: *Correa et al.* 1990; *Robertson,* 1968; *Svendsen,* 1969; 1974). Overfeeding in the dry period can induce hyperglycaemia in early lactation (*Holtenius et al.* 1996). Cows with LDA frequently show high plasma levels of glucose and insulin (*Muylle et al.* 1990; *DeCupere et al.* 1991). In man hyperglycaemia impairs gastric motility. Hyperinsulinemia per se has effects similar to hyperglycaemia and may be a mediator of the effects of hyperglycaemia (*Abrahamsson,* 1995). In a study with young heifers duodenal digesta flow was decreased after intravenous infusions of both glucose and insulin. It was suggested that the decreased digesta flow was due to elevated plasma level of insulin (*Meirhaeghe et al.* 1988).

The hormone glucagon is released from the pancreas in response to absorbed amino acids but also due to stress (see *Hedge et al.* 1987). In monogastric species and also in preruminant calves glucagon inhibits gastric motility (*Johansson & Segerström,* 1972; *McLeay & Bell* 1980).

One aim of the present study was to investigate the effects of an elevated plasma level of glucose, induced by an intravenous glucose infusion, on the abomasal emptying rate and the abomasal motility in dairy cows. We also studied the effects of an intravenous infusion of glucagon on those parameters in dairy cows.

Material and methods

Four cows of the Swedish red and white breed were used in the study. Three of the cows were dry, non pregnant, primiparous and one was in the first month of her second lactation. The weight of the cows ranged from 490-580 kg. The cows were fitted with a cannula (id 10 mm) in the abomasum about 30 cm from the pylorus (fig. 1).The cannula was made from polyvinylchloride with internal and external flexible flanges of polyethylene. Before the operation feed was withheld for 24 h. The operation was made with the animal in a standing position. After a paravertebral anaesthesia of the last thoracic and the first three lumbar nerves, an incision was made through the right abdominal wall behind the ventral half of the costal arch. The pyloric part of the abomasum was grasped with 2 sponge forceps and double purse string sutures were placed between the 2 forceps about 30 cm from the pylorus. Inside the sutures an incision was made. The barrel of the cannula, fitted with the internal flexible flange, was then gently pushed into the abomasal lumen through the incision inside the purse string sutures and secured with the sutures. The barrel was then exteriorized in a position ventrally to the incision in the abdominal wall with the aid

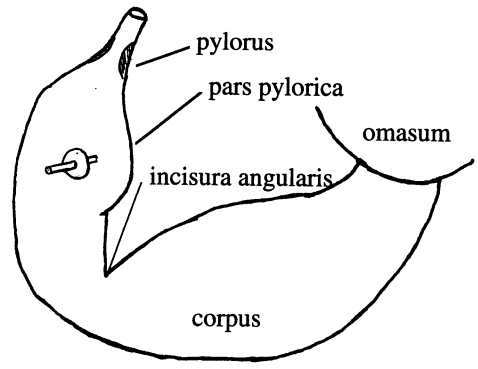

Figure 1. Location of the abomasal cannula.

of a sharp cone fitted to the barrel. Thereafter the cone was removed and the external flange, a locking nut and a cup were mounted. The peritoneum, internal and external muscle layers and the skin was closed as they are in routine flank laparatomy. The surgery was performed at least 1 month before the experimental series began. The dry cows were fed hay and concentrate twice daily at 07.00 and 15.00 to meet their requirements for maintenance. The lactating cow was fed hay and concentrate according the requirements for maintenance and milk production. The concentrate was given four times daily.

Experiment I

Glucose infusion experimental series: On the day of experiment at 09.00 feed and water was withdrawn and the cows were fitted with catheters in both jugular veins for infusion and blood sampling. An open-ended catheter was placed in the abomasum via the cannula, ending about 40 cm from the pylorus. The catheter was slowly perfused with water in order to avoid occlusion. A pressure transducer was connected to the catheter and the pressure changes were recorded continuously during the experiment on an ABB Goerz SE 462 scriptor, Wienna, Austria. Each experiment began with a control study. At 10.00h samples from the abomasal fluid and blood was taken. Three g of Cobalt-EDTA dissolved in 60 ml of water was then infused in the abomasum. Cobalt-EDTA, which has been shown to be a suitable marker for gastro-intestinal fluid flow, was prepared according to Udén et al. (1980). Samples from abomasal fluid, about 10 ml, were collected 18, 25, 32, 39, 46, 53 and 60 min after the Cobalt-EDTA infusion. The abomasal samples were immedeately centrifuged and pH measured in the supernatant fraction which then was frozen at -20 °C until analysis. Blood was collected 30 and 60 min after the abomasal Cobalt-EDTA infusion. All blood samples were centrifuged within 60 min and the plasma separated and frozen at –20 °C until they were analysed. Immediately after the 60 min samples was collected another, similar, dose of cobalt-EDTA was infused into the abomasum. At the same time a bolus dose of glucose, 60mg/kg body weight, were given intravenously via the jugular catheter contralateral to that used for blood sampling. The concentration of the glucose solution was 500 mg/ml. The bolus dose was followed by a continuous intravenous infusion of glucose (4 mg/kg bw/min) for 1 h. Samples from the abomasal fluid and blood was collected in an identical way during the glucose infusion as during the control period, without infusion.

Experiment II

The experimental series with glucagon followed the same protocol as the glucose experimental series with the exception that instead of glucose each cow got a bolus dose of glucagon (1 mg, Novo Nordisk, Bagsværd, Denmark) followed by a continuous intravenous infusion of glucagon (30pg/kg bw/min) during 1 h. One mg of glucagon was dissolved in 57 ml of 0.9% saline to which was added 3 ml blood in order to minimise binding of glucagon to tubings and syringes.

Analyses

Cobalt in the abomasal fluid was analysed in an atomic absorption spectrophotometer (Perkin-Elmer 4000, Norwalk, CT, USA). Assuming that the abomasal fluid outflow follows first-order kinetics the half life of fluid in abomasum could be estimated from the slope when the log concentration of cobalt was plotted versus time (*Gregory et al.* 1985). The turnover time of abomasal fluid could then be calculated as the half life / ln2. The calculation of the frequency of abomasal pressure changes was based on the number of distinct peaks during 5 min in the

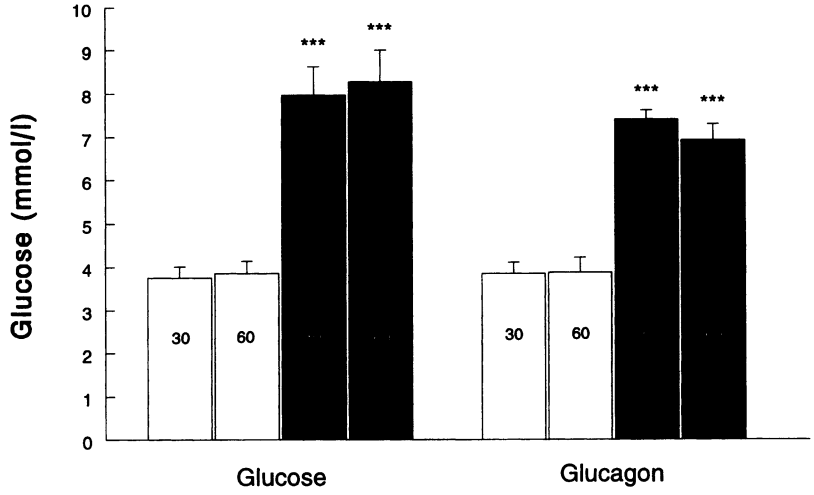

Figure 2. The plasma level of glucose in 4 cows. Blood was collected after 30 and 60 min of the control period and the infusion period respectively. Open bars represent the control period and the shaded bars represent periods with infusion of glucose and glucagon respectively. Data are given as means with standard errors.
*** Statistically significant difference versus the related time of the control ($p < 0.001$).

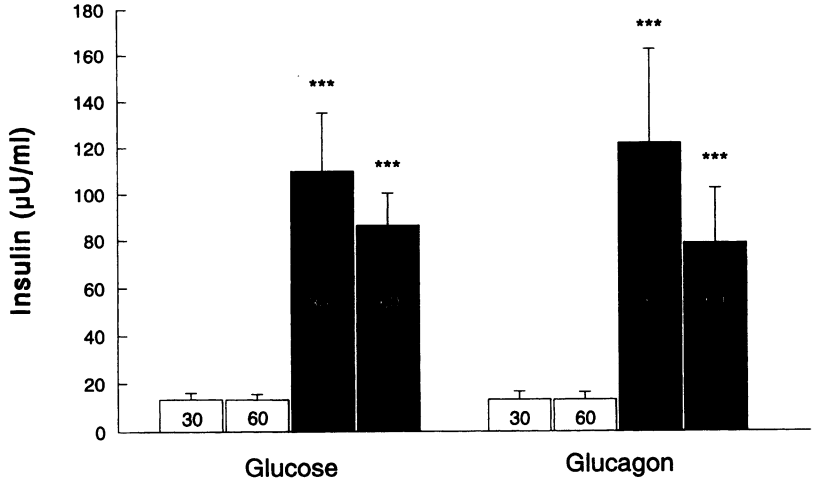

Figure 3. The plasma level of insulin in 4 cows. Blood was collected after 30 and 60 min of the control periods and the infusion periods respectively. Open bars represent the control periods and the shaded bars represent periods with infusion of glucose and glucagon respectively. Data are given as means with standard errors.
*** Statistically significant difference versus the related time of the control ($p < 0.001$).

Figure 4. The mean turnover time of abomasal fluid in the abomasum in 4 cows. The open bars represent the control period and the shaded bars represent period with infusion with glucose or glucagon respectively. Data are given as means with standard errors. **; *** Statistically significant difference versus the control ($p<0.01$; and $p<0.001$ respectively).

middle of each control period and also in the middle of each infusion period. The amplitude was estimated as the mean height above the basal level of all distinct peaks during the same five minutes period as when the frequency was calculated.

The concentration of glucose in plasma was analysed by enzyme technique using a commercial kit (Periochrom, Boehringer Mannheim Scandinavia, AB, Bromma, Sweden). Insulin in plasma was analysed using a commercial assay kit (Pharmacia RIA 100, Pharmacia Diagnostics AB, Uppsala, Sweden). pH in abomasal fluid was measured with glass electrode connected to a pH meter.

Values are presented as the mean ± standard error of the mean. Differences in abomasal fluid turnover time were tested by means of paired, 2-tailed, t-test. The significanse of differences in abomasal fluid pH were tested by ANOVA with repeated measurements using the general linear model procedure of Minitab Statistical Software (Release 11, Statistical Software, Birmingham, UK).

Results

Due to the glucose infusion plasma glucose rose to a level about 4 mmol/l above the basal level while the glucagon infusion induced an increase of the plasma level of glucose with about 3 mmol/l above the basal level. (Fig. 2). The plasma insulin did also show a striking increase after both glucose and glucagon infusion (Fig. 3).

As compared with the control period the turnover time of abomasal fluid increased with about 70% and 130% during glucose and glucagon infusion respectively (Fig. 4). The registrations of abomasal pressure changes during the control hour showed a pattern with periods with mainly small changes in pressure followed by periods with higher amplitude of the pressure

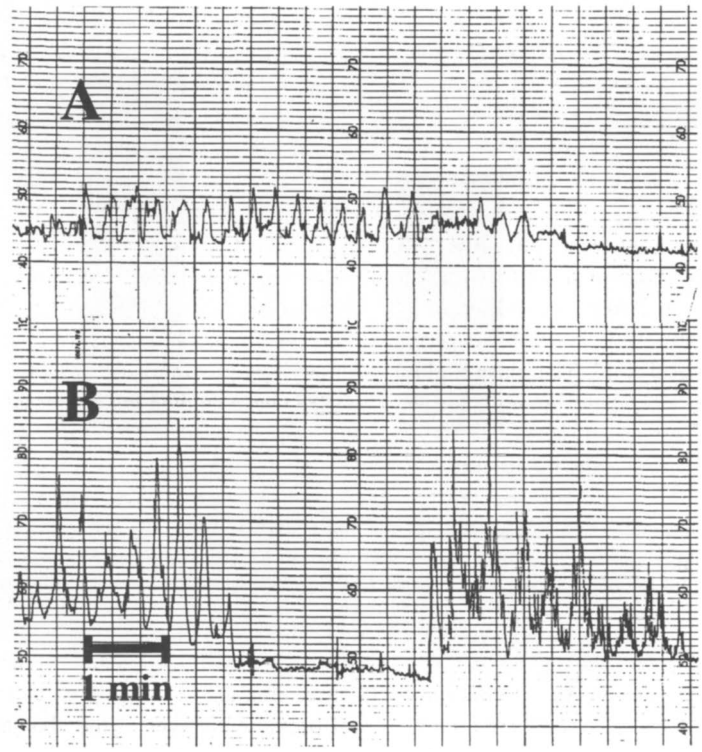

Figure 5. Two sections of data demonstrating changes in the intraluminal pressure in the abomasum of a cow during a control period (A) and during an intravenous glucose infusion (B).

changes in all cows. The frequency of the abomasal pressure changes during the periods with higher activity was 4.0±0.2 peaks / min and 4.1±0.2 for the control period prior to glucose and glucagon infusion respectively. In Fig. 5 the pattern of abomasal pressure changes before and during the glucose infusion is shown for one of the cows. The infusion induced periods with higher amplitude of the pressure changes than were observed during the control period (48±12 mm *versus* 15±3 mm $p<0.05$) in all cows. The frequency of the pressure changes was not significantly affected by the glucose infusion (3.9±0.1 peaks / min). Between the periods with large changes in the abomasal pressure there were periods with low activity which virtually were similar with the low activity periods during the control period. Glucagon infusion did not induce a clear change in the amplitude of abomasal pressure changes compared with the control period 16±4 mm vs 15±2 mm ns). The frequency of the abomasal pressure changes during the periods with higher amplitude was 4.1±0.2 peaks/min.

The average abomasal pH was 2.1±0.02 both during the control hour and during the glucose infusion. The glucagon infusion gave rise to a small but significant decrease in abomasal pH (2.1 ± 0.2 *versus* 1.9 ± 0.2 $p<0.01$).

Discussion

The turnover time of abomasal fluid were about 15 min in both of the two control measurements. The result is in agreement with the findings of *Jones & Poulsen* (1974), who injected a radioactice non-absorbable marker into the abomasum of 11 apparently healthy cows and measured the radioactivity externally. A recalculation of their data show that the mean turnover time of abomasal fluid also was 15 min.

The glucose infusion gave rise to a slower turnover time of abomasal fluid. The plasma glucose level increased with about 4 mmol/l during the infusion. A corresponding increase has been shown in dairy cows soon after parturition (*Church,* 1979). Thus, the increase in the plasma glucose level must be regarded as within physiological limits. In a previous study with young heifers an intravenous glucose load gave rise to a delayed abomasal outflow, however the increase in plasma glucose was much higher in that study (*Meirhaeghe et al.* 1988). To the best of our knowledge there are no previous reports published which show that an elevation of plasma glucose, within physiological limits, delays the turnover of abomasal fluid in cattle. In man hyperglycaemia delays gastric emptying and the degree of inhibition is related to the glucose level in blood (*MacGregor et al.* 1976; *Øster-Jörgensen et al.* 1990). It has been suggested that the glucose-induced hyperinsulinaemia may be a mediator of the impaired gastric emptying in man as well as in cattle although other mechanisms also seem to be essential for the hyperglycaemic depression of gastrointestinal motility (*Abrahamsson,* 1995; *Meirhaeghe et al.* 1988). In the present study the plasma insulin level, as expected, rose during the glucose infusion. Thus, it cannot be excluded that the slower turnover time of abomasal fluid was induced by the hyperinsulinaemia. On the other hand, the abomasal outflow was remarkably increased despite a short initial inhibition during insulin infusion (1U/kg body weight/hour) in milk fed calves (*McLeay & Bell* 1980). The authors suggested that the stimulatory effect was due to the insulin-induced hypoglycaemia.

Although the glucose infusion reduced the abomasal rate of fluid outflow the amplitude of the pressure changes was increased. The result is in agreement with the observation of *Vlaminck et al.* (1984a) that pressure changes in the abomasum were not correlated to the abomasal emptying rate in cattle. *Kuiper & Breukink* (1988) have studied the abomasal myo-electrical activity of adult cattle. Beginning at the *insisura angularis* bursts of spiking activity, leading to contractions, were propagating towards the *pylorus.* The bursts of spiking activity showed a regular pattern with a frequency of about 4 per min. Between the periods of regular bursts of spiking activity there were periods with relative myo-electrical inactivity. It is interesting that in the present study the pattern of pressure changes in the abomasum showed striking similarities with the myo-electrical activity in the same region of the abomasum as shown by *Kuiper & Breukink* (1988). On the other hand, in a study of *Vlaminck et al.* (1984a) there was no correlation between abomasal myo-electrical activity and abomasal pressure changes in cattle. During glucose infusion periods with higher amplitude of the pressure changes were observed in *pars pylorica.* In man hyperglycaemia, induced by an intravenous glucose infusion, gave rise to a stimulation of the pressure waves in the pyloric region (*Fraser et al.* 1991). These authors suggested that such a stimulation may be part of an abnormal gastric motility which in turn gives rise to the delayed gastric emptying often observed in hyperglycaemic diabetic patients. In cattle a slow emptying rate can occur in periods with an intensive myo-electric activity in the pyloric region (*Vlaminck et al.* 1984b). However, it has

been suggested that in contrast to monogastric species, the pyloric sphincter plays a regulating role in the gastric emptying process of the ruminants and that the motility of *pars pylorica* is of less importance for this process (*Ehrlein,* 1970; *Vlaminck et al.* 1984a). Thus, the delayed turnover of abomasal fluid and the altered pattern of intraabomasal pressure changes may have different causes.

In the present study turnover of abomasal fluid was substantially delayed during the glucagon infusion. Glucagon has been shown to inhibit gastric emptying in monogastric species (*Jonderko et al,* 1988; *Johansson & Segerström,* 1972) and abomasal emptying in preruminant calves (*McLeay & Bell* 1980). To the best of our knowledge the effects of glucagon on abomasal emptying in adult cattle has not been published previously. The glucagon infusion induced an endogenous glucose secretion and a concomitant rise in insulin as expected. It could not be excluded that the observed inhibiting effect of glucagon on abomasal emptying rate was mediated by the increased plasma levels of glucose and insulin. However, in contrast to the glucose infusion glucagon did not induce periods with increased pressure changes in *pars pyloricum.* The glucose level was slightly lower during the glucagon infusion as compared to the glucose infusion. It can not be excluded that a higher plasma glucose level was needed in order to induce an altered pattern of intraabomasal pressure changes.

The pH of the abomasal fluid showed a small but significant drop due to the glucagon infusion. The result is intriguing since glucagon inhibits gastric acid secretion in both monogastric species and in preruminating calves (*Clarke et al.* 1960; *Loud et al.* 1988; *McLeay & Bell* 1980). It is possible that the decreased abomasal pH reflects a decreased inflow of digesta from the reticulo-rumen and not an increased HCl secretion. The pH of the digesta in the reticulo-rumen is normally kept between 6 and 7. In adult sheep in which the endogenous glucagon secretion was stimulated by alanine the abomasal outflow was depressed but the abomasal pH was not significantly affected (*Holtenius & Björnhag* 1994). I thus seems reasonable to assume that glucagon does not have the same inhibiting effect on gastric acid secretion in adult ruminants as in monogastric species.

The results of the present study indicate a disturbed abomasal function in cattle with hyperglycaemia. It remains to be investigated if it is a direct effect of the hyperglycaemia or if it is secondary to the elevated insulin level. It is not unlikely that hyperglycaemia is a significant factor involved in the pathogenesis of displaced abomasum.

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Sammanfattning

Effekter av intravenös infusion av glukos och pankreas-glukagon på löpmagsfunktionen hos mjölkkor.

Två experiment utfördes där inverkan av en intravenös glukosinfusion respektive intravenös infusion av glukagon på löpmagsfunktionen studerades på kor. Plasmahalten av glukos och glukagon registrerades också. Fyra kor av svensk rödbrokig ras, försedda med fistelinsats i löpmagen, användes i experimenten. I glukosinfusionsförsöket erhöll de fyra

korna, efter en inledande kontrollperiod på 60 min utan behandling, en intravenös glukosinfusion (4 mg/kg kroppsvikt/min.) också under 60 min. Under glukosinfusionen steg glukoshalten med ungefär 4 mmol/l. Den förhöjda glukoshalten inducerade en markerad frisättning av insulin. Omsättningstiden för ingesta i löpmagen ökade från 15,7±1,2 min till 27,8±3,5 min ($p<0,01$) under glukosinfusionen. Amplituden av tryckförändringarna i löpmagen mer än fördubblades under glukosinfusionen ($p<0,05$) men frekvensen av tryckförändringarna påverkades inte.

Samma försöksuppläggning som under glukos försöket användes under glukagonförsöket men korna erhöll då i stället en intravenös infusion av glukagon (30pg/kg kroppsvikt). Glukagoninfusionen ledde till en frisättning av glukos vilket höjde plasmahalten med omkring 3 mmol/l. Insulinnivån i plasma steg ungefär lika mycket som under glukosinfusionen. Omsättningstiden för ingesta i löpmagen ökade från 15,4±1,7 min till 34,8±1,9 min under glukagoninfusionen ($p<0,001$). Varken frekvensen eller amplituden av förändringarna i löpmagstrycket påverkades dock av glukagoninfusionen. Försöken tyder på att hyperglykemi stör löpmagsfunktionen hos kor. Det återstår att undersöka om de observerade effekterna på löpmagen var en direkt effekt av den förhöjda plasmahalten av glukos eller en indirekt effekt av den förhöjda insulinnivån.

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